Post Cardiac Arrest Management

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Chair – “Evidence-based guideline: Reducing brain injury following cardiopulmonary resuscitation”
American Academy of Neurology – under peer review

Member – AHA-ILCOR 2015 CPR Guideline Review Panel
Writing Panel – Post Arrest Chapter of 2015 CPR Guidelines
Science Subcommittee member, ECC AHA (CPR Guidelines)

Immediate Past President /
Chair, Global Partners – Neurocritical Care Society
Objectives:

• Review Post Cardiac Arrest Syndrome: brain injury
• Impact of temperature on brain injury
• Clinical studies of temperature management after CPR
• Existing guidelines and upcoming considerations

Dr. C. Park, Anesthesia Resident, Baltimore City Hospital; Capt. Martin McMahon, Chief, Baltimore Fire Department Ambulance Service and Dr. Peter Safar, Chief, Department of Anesthesia, Baltimore City Hospital, performing one of the earliest resuscitation studies using CPR.

ABCs of resusciation

Baltimore City Hospitals 1950s-1960 (now Johns Hopkins Bayview Medical Center)
Post–Cardiac Arrest Syndrome
Epidemiology, Pathophysiology, Treatment, and Prognostication
A Consensus Statement From the International Liaison Committee on Resuscitation (American Heart Association, Australian and New Zealand Council on Resuscitation, European Resuscitation Council, Heart and Stroke Foundation of Canada, InterAmerican Heart Foundation, Resuscitation Council of Asia, and the Resuscitation Council of Southern Africa); the American Heart Association Emergency Cardiovascular Care Committee; the Council on Cardiovascular Surgery and Anesthesia; the Council on Cardiopulmonary, Perioperative, and Critical Care; the Council on Clinical Cardiology; and the Stroke Council

Endorsed by the American College of Emergency Physicians, Society for Academic Emergency Medicine, Society of Critical Care Medicine, and Neurocritical Care Society

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Post–Cardiac Arrest Syndrome

- Post–cardiac arrest brain injury
  - Systemic ischemia-reperfusion response
  - Persistent precipitating pathology
- Post–cardiac arrest myocardial dysfunction
Post Cardiac Arrest Syndrome
Therapeutic Strategies

- Goal Directed Therapy
- Early Hemodynamic Optimization
- Oxygenation
- Ventilation
- Circulatory Support
- Management of ACS

**Therapeutic hypothermia**
Cardiac Arrest and Death

**Figure 47.2.** Mortality/survival and cause of death in comatose survivors of cardiac arrest (Brain Resuscitation Clinical Trial I [BRCT I], n = 242; Brain Resuscitation Clinical Trial II [BRCT II], n = 516).

**Functional Outcome CPR - Survivors**

Edgren et al 1989
Brain Injury after Global Ischemia

Brain Selective Vulnerability
- CA-1 hippocampus
- Neocortex cell layers

Brain Relative Vulnerability
- Thalamus
- Cerebellum (Purkinje cells
- Putamen and Caudate

Relative Tolerance to Ischemia
- Brainstem
Global Ischemia

Acute Presentation

Brain Death
Coma/PVS
Stuporous/Delirious
Cognitive Deficit
Seizures (cortex only)

Problem: Multi-systems

Cortex
Subcortex
Thalamus
Upper Brainstem
Graveyard of Clinical Trials: Neuroprotection in Global Cerebral Ischemia

**Thiopental LD - no benefit**
BRCT1 (NEJM, 1986) n=262

**Glucocorticoid - associated with complications**
BRCT1 (JAMA, 1989) n=262

**Nimodipine – no benefit**
Roine, et al (JAMA, 1990) n=748

**Lidoflazine - no benefit**
BRCT 2 (NEJM, 1991) n=520

**Non-Glucose IV Fluid – no benefit**
Longstreth, et al (Neurology 1993) n=748

**Magnesium/Diazepam – no benefit**
Longstreth, et al (Neurology 2002) n=300

**Vasopressin - no neuro benefit**
Vasopressin–OHA V-Fib Arrest (Lancet, 2001) n=40
Canadian Vasopressin - Epi Study (Lancet, 2001) n=200

Except for IV-TPA no drug improves outcome in brain ischemia!!
And then there was

...THERAPEUTIC HYPOTHERMIA
Injury Mechanism

Box 1
Mechanisms of anoxic-ischemic brain injury

Immediate
1. Cellular energy depletion, with anaerobic metabolism
2. Collapse of transmembrane sodium and potassium gradients
3. Failure of synaptic transmission, axonal conduction, and action potential firing
4. Intracellular acidosis
5. Hypercalcemia
6. Glutamate release, with neuronal hyperexcitability
8. Mitochondrial dysfunction
9. Reperfusion, with generation of reactive oxygen species and lipid peroxidation
10. Elevated production of nitric oxide and peroxynitrite
11. Blood-brain barrier dysfunction
12. Loss of cerebral autoregulation

Delayed
1. Release of proinflammatory mediators (e.g., tumor necrosis factor-α and interleukin-1)
2. Inflammatory cells recruitment
3. Complement activation
4. Caspase activation with apoptosis
5. Coagulation activation

Data from Refs. 27, 133–135

Box 2
Protective mechanism of therapeutic hypothermia

Early
1. Decrease of cerebral metabolism
2. Decrease in mitochondrial injury and dysfunction
3. Improve ion pump function, decrease intracellular influx of calcium
4. Improve cell membrane leakage, decrease intracellular acidosis
5. Decrease production of reactive oxygen species
6. Decrease formation of cytotoxic edema

Late
1. Decrease of local production of endothelin and thromboxane A2, increase generation of prostaglandins
2. Improve tolerance for ischemia
3. Decrease neuroinflammation
4. Decrease apoptosis
5. Decrease cerebral thermo-pooling
6. Decrease vascular permeability
7. Activation of protective genes
8. Suppression of cortical spreading depression
9. Suppression of seizure activity
10. Decrease coagulation activation and formation of microthrombi

Data from Refs. 71, 80
Keep in Mind:

Cardiac Rhythms/Place of Arrest: Markers of Severity - Not effectiveness of TX

Place of Arrest:
OOHCA – Generally healthier
IHCA – Sicker pts in hospital

Cardiac Rhythms:
Shockable (pulseless VT/VF)
Non-Shockable Rhythms (PEA/Asystole)
Outcomes: Cardiac Rhythms & Cardiac Arrest

Ultimately all malignant arrhythmias will deteriorate to asystole

V. Fibrillation
Better outcome
Shorter Arrest Duration
Less co-morbidity

Asystole
Poor Outcome
Longer Arrest Duration
More co-morbidity

Which rhythm results in more brain injury?
N=275 of 3551
OHA - ROSC – VF/VT
CA 5-15m/CPR<60m
RCT-Consent Waived
Blinded Outcome Assessment

N=138

Hypothermia

Treatment
External cooling (in 4 hours)
32-34°C for 24 hours
Sedated (fentanyl/midaz)
Paralyzed prn shivers
Start at ED to ICU
Passive rewarm (8h after)

1° Outcome
Discontinued Early (n=14)
Death
Complication Protocol issues

2° Outcome
6 month Mortality
Complication Rates

Normothermia
N=137

Treatment
Sedated/Paralyzed
Normothermia: 37°C

1° Outcome
PCPC Categories
(1,2 - good & 3,4,5 – poor)

2° Outcome
6 month Mortality
Complication Rates

Hypothermia

1° Outcome
PCPC Categories
(1,2 - good & 3,4,5 – poor)

2° Outcome
6 month Mortality
Complication Rates

HACA Study Group

NEJM 2002;346 (8) 549-56

### Table 2. Neurologic Outcome and Mortality at Six Months.

<table>
<thead>
<tr>
<th>Outcome</th>
<th>Normothermia</th>
<th>Hypothermia</th>
<th>Risk Ratio (95% CI)</th>
<th>P Value†</th>
</tr>
</thead>
<tbody>
<tr>
<td>no./total no. (%)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Favorable neurologic outcome†</td>
<td>54/137 (39)</td>
<td>75/136 (55)</td>
<td>1.40 (1.08–1.81)</td>
<td>0.009</td>
</tr>
<tr>
<td>Death</td>
<td>76/138 (55)</td>
<td>56/137 (41)</td>
<td>0.74 (0.58–0.95)</td>
<td>0.02</td>
</tr>
</tbody>
</table>
Induced Hypothermia

Bernard, et al NEJM 2002;346 (8) 557-63

Even days
Normothermia
Start at field
N=34

Treatment
Sedated/Paralyzed
Normothermia: 37°C

Outcome
Discharge to home
Rehab facility
Nursing home
Death

Odd days
Hypothermia
Started at field
Cold Packs
N=43

Treatment
External cooling
33°C for 12 hours
Mid/vec prn
Start at ED to ICU
Active rewarm (18h)

Outcome
Discharge to home
Rehab facility
Nursing home
Death

N=77
OHA - ROSC - VFIB
Coma - ED in 1-4 hrs
Induced Hypothermia
Bernard, et al NEJM 2002;346 (8) 557-63

Patient Outcome (Bernard, et al 2002)

Survivors

Death
Withdrawal of life support
Brain death
Death: Cardiac failure

Hypothermia: 49% Normothermia: 26%
(95%CI: 13-43, p=0.046)
Therapeutic Hypothermia
Complications after CA (0-7 days)

Complication after CA (0-7 Days)
NEJM (European Hypothermia Study after CA) 2002

- 22% more complications in H than N (NS)
- Pneumonia (NNH=12)
- Bleeding (NNH=14)
- Sepsis (NNH=16)

Any Bleeding
Plt Transfusions
Pneumonia
Sepsis
Pancreatitis
Renal Failure
Hemodialysis
Seizure
Arrhythmia*
A comprehensive, structured, multidisciplinary system of care should be implemented in a consistent manner for the treatment of post–cardiac arrest patients (Class I, LOE B). Programs should include as part of structured interventions therapeutic hypothermia; optimization of hemodynamics and gas exchange; immediate coronary reperfusion when indicated for restoration of coronary blood flow with percutaneous coronary intervention (PCI); glycemic control; and neurological diagnosis, management, and prognostication.
In summary, we recommend that comatose (ie, lack of meaningful response to verbal commands) adult patients with ROSC after out-of-hospital VF cardiac arrest should be cooled to 32°C to 34°C (89.6°F to 93.2°F) for 12 to 24 hours (Class I, LOE B).

Induced hypothermia also may be considered for comatose adult patients with ROSC after in-hospital cardiac arrest of any initial rhythm or after out-of-hospital cardiac arrest with an initial rhythm of pulseless electric activity or asystole (Class IIb, LOE B).
Non-shockable Rhythms: Mixed signal but not harmful

Is Hypothermia After Cardiac Arrest Effective in Both Shockable and Nonshockable Patients? Insights From a Large Registry
Florence Dumas, David Grimaldi, Benjamin Zuber, Jérôme Fichet, Julien Charpentier, Frédéric Pène, Benoît Vivien, Olivier Varenne, Pierre Carli, Xavier Jouven, Jean-Philippe Empana and Alain Cariou

Clinical paper
Therapeutic hypothermia is associated with improved neurologic outcome and survival in cardiac arrest survivors of non-shockable rhythms
Justin B. Lundbye, Mridula Rai, Bhavadharini Ramu, Ali Reza Hosseini-Khalili, Dadong Li, Hanna B. Slim, Sanjeev P. Bhavnani, Sanjeev U. Nair, Jeffrey Kluger

Clinical paper
Mild therapeutic hypothermia is associated with favourable outcome in patients after cardiac arrest with non-shockable rhythms
Christoph Testori, Fritz Sterz, Wilhelm Behringer, Moritz Haugk, Thomas Uray, Andrea Zeiner, Andreas Janata, Jasmin Arrich, Michael Holzer, Heidrun Losert

Many more papers....
Study objective: To compare two target temperatures, both intended to prevent fever.

International RCT: 950 unconscious adults after out-of-hospital cardiac arrest of presumed cardiac cause to targeted temperature management at either 33° C or 36° C.

The primary outcome: all-cause mortality to end of the trial.

Secondary outcomes: composite of poor neurologic function (CPC and mRS) or death at 180 days
Design and timeline  (modified from N Nielsen)

Inclusion Criteria
- Age >= 18 years
- Out-of-hospital cardiac arrest of presumed cardiac cause
- Unconsciousness (Glasgow Coma Score <8) after sustained return of spontaneous circulation (ROSC) (20 minutes of circulation)

Cooling device with feedback control; surface and endovascular mixed
Body Temperature during the Intervention Period.


Probability of Survival through the End of the Trial.

No. at Risk

<table>
<thead>
<tr>
<th>Temperature</th>
<th>At Risk</th>
<th>Days since Randomization</th>
</tr>
</thead>
<tbody>
<tr>
<td>33°C group</td>
<td>473</td>
<td>230 151 64 15</td>
</tr>
<tr>
<td>36°C group</td>
<td>466</td>
<td>235 144 68 12</td>
</tr>
</tbody>
</table>

P = 0.51

Table 2. Outcomes.

<table>
<thead>
<tr>
<th>Outcome</th>
<th>33°C Group no./total no. (%)</th>
<th>36°C Group no./total no. (%)</th>
<th>Hazard Ratio or Risk Ratio (95% CI)*</th>
<th>P Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Primary outcome: deaths at end of trial</td>
<td>235/473 (50)</td>
<td>225/466 (48)</td>
<td>1.06 (0.89–1.28)</td>
<td>0.51</td>
</tr>
<tr>
<td>Secondary outcomes</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Neurologic function at follow-up†</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>CPC of 3–5</td>
<td>251/469 (54)</td>
<td>242/464 (52)</td>
<td>1.02 (0.88–1.16)</td>
<td>0.78</td>
</tr>
<tr>
<td>Modified Rankin scale score of 4–6</td>
<td>245/469 (52)</td>
<td>239/464 (52)</td>
<td>1.01 (0.89–1.14)</td>
<td>0.87</td>
</tr>
<tr>
<td>Deaths at 180 days</td>
<td>226/467 (48)</td>
<td>220/466 (47)</td>
<td>1.01 (0.87–1.15)</td>
<td>0.92</td>
</tr>
</tbody>
</table>

* The hazard ratio is shown for the primary outcome, and risk ratios are shown for the secondary outcomes. CI denotes confidence interval.
† The neurologic follow-up was specified in the protocol to be performed at 180 days ± 2 weeks, but the time to follow-up was in some cases several weeks longer for logistic reasons. The Cerebral Performance Category (CPC) scale ranges from 1 to 5, with 1 representing good cerebral performance or minor disability, 2 moderate cerebral disability (function is sufficient for independent activities of daily life), 3 severe cerebral disability, 4 coma or vegetative state, and 5 brain death. Scores on the modified Rankin scale range from 0 to 6, with 0 representing no symptoms, 1 no clinically significant disability despite some symptoms, 2 slight disability (patient is able to look after own affairs without assistance), 3 moderate disability (patient requires some help but is able to walk unassisted), 4 moderately severe disability (patient is unable to attend to own bodily needs), 5 severe disability (patient is bedridden), and 6 death.

Table 3. Neurologic Scores,*

<table>
<thead>
<tr>
<th>Variable</th>
<th>33°C Group</th>
<th>36°C Group</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total no. of patients</td>
<td>469</td>
<td>464</td>
</tr>
<tr>
<td>Category — no. (%)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>1</td>
<td>195 (42)</td>
<td>183 (39)</td>
</tr>
<tr>
<td>2</td>
<td>23 (5)</td>
<td>39 (8)</td>
</tr>
<tr>
<td>3</td>
<td>17 (4)</td>
<td>20 (4)</td>
</tr>
<tr>
<td>4</td>
<td>6 (1)</td>
<td>2 (0.5)</td>
</tr>
<tr>
<td>5</td>
<td>228 (49)</td>
<td>220 (47)</td>
</tr>
<tr>
<td>P value for trend</td>
<td>0.85</td>
<td></td>
</tr>
<tr>
<td>Best, or lowest numerical, CPC during trial</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Total no. of patients</td>
<td>472</td>
<td>466</td>
</tr>
<tr>
<td>Category — no. (%)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>1</td>
<td>209 (44)</td>
<td>205 (44)</td>
</tr>
<tr>
<td>2</td>
<td>25 (5)</td>
<td>41 (9)</td>
</tr>
<tr>
<td>3</td>
<td>37 (8)</td>
<td>37 (8)</td>
</tr>
<tr>
<td>4</td>
<td>201 (43)</td>
<td>183 (39)</td>
</tr>
<tr>
<td>5</td>
<td>NA</td>
<td>NA</td>
</tr>
<tr>
<td>P value for trend</td>
<td>0.89</td>
<td></td>
</tr>
<tr>
<td>Modified Rankin scale score at follow-up†</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Total no. of patients</td>
<td>469</td>
<td>464</td>
</tr>
<tr>
<td>Score — no. (%)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>0</td>
<td>88 (19)</td>
<td>89 (19)</td>
</tr>
<tr>
<td>1</td>
<td>69 (15)</td>
<td>83 (18)</td>
</tr>
<tr>
<td>2</td>
<td>50 (11)</td>
<td>34 (7)</td>
</tr>
<tr>
<td>3</td>
<td>17 (4)</td>
<td>19 (4)</td>
</tr>
<tr>
<td>4</td>
<td>8 (2)</td>
<td>11 (2)</td>
</tr>
<tr>
<td>5</td>
<td>9 (2)</td>
<td>8 (2)</td>
</tr>
<tr>
<td>6</td>
<td>228 (49)</td>
<td>220 (47)</td>
</tr>
<tr>
<td>P value for trend</td>
<td>0.67</td>
<td></td>
</tr>
</tbody>
</table>

* P values for trend were calculated with the use of the Cochran–Armitage test. NA denotes not applicable.
† The neurologic follow-up was specified in the protocol to be at 180 ± 14 days, but the time to follow-up was in some cases several weeks longer for logistic reasons.
Good Outcomes Comparison: 3 Trials

HACA 2002  Bernard 2002  Nielsen 2002
2 liters of 4°C IV bolus followed by standard of care at hospital
**re-arrest in the treatment group (26% vs 21%, p=0.008)
lower O2 saturation (PaO2 189 vs 218, p<0.001),
pulmonary edema on CXR (41% vs 30%, p<0.001)
• “…Pending formal Consensus on the optimal temperature, we suggest that clinicians provide postresuscitation care based on the current treatment recommendations (ILCOR/AHA). We accept that some clinicians may make a local decision to use a target temperature of 36°C pending this further guidance.”
2015 Recommendations—Updated

We recommend that comatose (ie, lack of meaningful response to verbal commands) adult patients with ROSC after cardiac arrest have TTM (Class I, LOE B-R for VF/pVT OHCA; Class I, LOE C-EO for non-VF/pVT (ie, “nonshockable”) and in-hospital cardiac arrest).

We recommend selecting and maintaining a constant temperature between 32°C and 36°C during TTM (Class I, LOE B-R).
“essentially no patients for whom temperature control somewhere in the range between 32°C and 36°C is contraindicated”

Specific features of the patient may favor selection of one temperature over another for TTM. Higher temperatures (~36°C) might be preferred in patients for whom lower temperatures convey some risk (eg, bleeding),

Lower temperatures (~32°C) might be preferred when patients have clinical features that are worsened at higher temperatures (eg, seizures, cerebral edema).

**caveat – lower temps (32°C) may be selected for worse neurologic injuries and result in the impression that it is less effective in practice**
Hypothermia in the Prehospital Setting

2015 Recommendation—New

We recommend against the routine prehospital cooling of patients after ROSC with rapid infusion of cold intravenous fluids (Class III: No Benefit, LOE A).

Whether different methods or devices for temperature control outside of the hospital are beneficial is unknown.
Avoidance of Hyperthermia

2015 Recommendation—New
It may be reasonable to actively prevent fever in comatose patients after TTM (Class IIb, LOE C-LD).

Fever in the post–cardiac arrest patient who is not treated with TTM is associated with poor outcome.

After rewarming to normothermia from TTM, fever occurs in a significant proportion of patients. Occurrence of hyperthermia during the first few days after cardiac arrest was associated with worse outcome in some studies.
Summary for TTM
I hope for now - 2014

OOHCA
VF/nonVF
Pre-hospital Cooling
No Benefit (?)harmful
Kim/Castren/Bernard

IHCA PEA/Asystole
TTM @ in-hospital
May be beneficial
Multiple studies
Class 2A-2B

IHA-CA (No RCT)
Multiple studies
Class 2B

OOHCA
Pulseless VT/VF
TTM @ in-hospital
Beneficial outcome/qol
HACA/Bernard/Neilsen
Class 1

OOHCA/ PEA/Asystole
Neilsen subgroup
Class 1(?)
Post Cardiac Arrest Syndrome
Therapeutic Strategies

Therapeutic hypothermia
- Sedation and Neuromuscular Blockade
- Seizure control
- Glucose Control
- Neuroprotection
Post Cardiac Arrest Syndrome Therapeutic Strategies

Prognostication

Continue Care

Withdrawal of life-supporting therapies
2015 Recommendation—New
Avoiding and immediately correcting hypotension (systolic blood pressure less than 90 mm Hg, MAP less than 65 mm Hg) during postresuscitation care may be reasonable (Class IIb, LOE C-LD).
2015 Recommendations—Updated
Coronary angiography should be performed emergently (rather than later in the hospital stay or not at all) for OHCA patients with suspected cardiac etiology of arrest and ST elevation on ECG (Class I, LOE B-NR).
Part 8: Post–Cardiac Arrest Care

2015 American Heart Association Guidelines Update for Cardiopulmonary Resuscitation and Emergency Cardiovascular Care

Clifton W. Callaway, Chair; Michael W. Donnino; Ericka L. Fink; Romergryko G. Geocadin; Eyal Golan; Karl B. Kern; Marion Leary; William J. Meurer; Mary Ann Peberdy; Trevonne M. Thompson; Janice L. Zimmerman

2015 Recommendation—Updated

The benefit of any specific target range of glucose management is uncertain in adults with ROSC after cardiac arrest (Class IIb, LOE B-R).
Part 8: Post–Cardiac Arrest Care

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Clifton W. Callaway, Chair; Michael W. Donnino; Ericka L. Fink; Romergryko G. Geocadin; Eyal Golan; Karl B. Kern; Marion Leary; William J. Meurer; Mary Ann Peberdy; Treveronne M. Thompson; Janice L. Zimmerman

2015 Recommendation—Updated
Maintaining the PaCO2 within a normal physiological range, taking into account any temperature correction, may be reasonable (Class IIb, LOE B-NR).

To avoid hypoxia in adults with ROSC after cardiac arrest, it is reasonable to use the highest available oxygen concentration until the arterial oxyhemoglobin saturation or the partial pressure of arterial oxygen can be measured (Class IIa, LOE C-EO).

When resources are available to titrate the FIO2 and to monitor oxyhemoglobin saturation, it is reasonable to decrease the FIO2 when oxyhemoglobin saturation is 100%, provided the oxyhemoglobin saturation can be maintained at 94% or greater (Class IIa, LOE C-LD).
2015 Recommendations—Updated
An EEG for the diagnosis of seizure should be promptly performed and interpreted, and then should be monitored frequently or continuously in comatose patients after ROSC (Class I, LOE C-LD).

The same anticonvulsant regimens for the treatment of status epilepticus caused by other etiologies may be considered after cardiac arrest (Class IIb, LOE C-LD).
All studies focused on prognostication of poor outcome
Patients NOT treated with hypothermia
Does neuro prognostication have an impact on patient care?

Survivors (17%)
Somatic Death (14%)
Death by withdrawal of life sustaining measures (69%)

All GCS ≤ 4 at time of decision to withdraw life sustaining therapy

Geocadin, et al 2006
Can we predict the outcome?

No intra-arrest factor is a reliable predictor of functional outcome

CPR quality/Arrest time/CPR time/Initial Rhythm/Temp; ETCO2; non-cardiac cause of arrest


Only in post arrest: Neuro exam
But not earlier than 3 days,
With TH ~5-7 days

No pre-cardiac arrest factor is a reliable predictor of functional outcome

Race/baseline health/lifestyle, etc

Post-Arrest Predictors
Major Confounders

- Hemodynamic instability, severe metabolic derangement and drugs may mask neurologic evaluation – error in prognosis

- Hypothermia patients – have delayed clearance of sedative and paralytics

- Neurologic recovery may be delayed by hypothermia

From Wijdicks, et al 2006
2015 Recommendations—New and Updated
In comatose patients who are not treated with TTM, the absence of pupillary reflex to light at 72 hours or more after cardiac arrest is a reasonable exam finding with which to predict poor neurologic outcome (FPR, 0%; 95% CI, 0%–8%; Class IIa, LOE B-NR).

In comatose patients who are treated with TTM, the absence of pupillary reflex to light at 72 hours or more after cardiac arrest is useful to predict poor neurologic outcome (FPR, 1%; 95% CI, 0%–3%; Class I, LOE B-NR).
2015 Recommendations—Updated
In comatose post–cardiac arrest patients who are treated with TTM, it may be reasonable to consider persistent absence of EEG reactivity to external stimuli at 72 hours after cardiac arrest, and persistent burst suppression on EEG after rewarming, to predict a poor outcome (FPR, 0%; 95% CI, 0%–3%; Class IIb, LOE B-NR).

Intractable and persistent (more than 72 hours) status epilepticus in the absence of EEG reactivity to external stimuli may be reasonable to predict poor outcome (Class IIb, LOE B-NR).
EMERGENCY NEUROLOGICAL LIFE SUPPORT (ENLS):
WHAT TO DO IN THE CRITICAL FIRST HOURS OF A NEUROLOGICAL EMERGENCY
http://www.neurocriticalcare.org/
Come join us in Washington DC
September 15-18, 2016

Save the Date

Contact: info@neurocriticalcare.org